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http://europepmc.org/articles/PMC3649201	Current Views on Genetics and Epigenetics of Cholesterol Gallstone ...	190	3	13	<1
http://onlinelibrary.wiley.com/doi/10.1111/j.1365-2796.2007.01783.x/full	Gallstone disease - Marschall - 2007 - Wiley Online Library	162	3	154	3
https://www.researchgate.net/publication/236925465_Current_Views_on_Genetics_and_Epigenetics_of_Cholesterol_Gallstone_Disease	Current Views on Genetics and Epigenetics of Cholesterol Gallstone ...	107	2	0	<1
https://www.researchgate.net/publication/13211823_Gallstone_disease_risk_in_relation_to_body_mass_index_and_waist-to-hip_ratio_in_Japanese_men	Gallstone disease risk in relation to body mass index and waist-to-hip ...	43	1	43	1
http://www.oalib.com/paper/3068171	Current Views on Genetics and Epigenetics of Cholesterol Gallstone ...	190	3	0	<1
http://www.researchgate.net/publication/11281703_Human_cholesterol_7-hydroxylase_(CYP7A1)_deficiency_has_a_hypercholesterolemic_phenotype	Human cholesterol 7α-hydroxylase (CYP7A1) deficiency has a ...	51	1	51	1
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INTRODUCTIONThere are numerous ways of practicing the healing art, but there is only one actual way. Natural remedies are the simple interventions of nature, that will not weaken the system through there powerful properties. A proper diet, pure air, pure water, cleanliness, purity of life and a strong trust in divine power are remedies for the want of which thousand are failing in their lives, yet these remedies are going out of date because there clever full use require so much time and work that the people do not appreciate. Many are living in violation of health and are ignorant of the relation between their habit of eating, drinking and working to sustain their health. Rising wages and growth in economy lead to substation of helpers or appliances for physical work, taking desk jobs instead of cultivation in the fields. These following factors also boost more sedentary life style such as watching TV and computer use and well of urban people travel by car instead of providing exercises to the body by walking. Prakash shetty of Food Association of India (FAO) says that food habits and lifestyle changes as economy develops. There is significance raise in the consumption of fat rich and junk food, high caloric drinks and energy dense foods due to their easy availability and less time consumption in preparing or purchasing. Urbanization puts junk foods and fast foods within easy reach of population. In India these factors make to the rise of bad eating, drinking habits and lack of physical exercises leads to various disorders especially gastrointestinal problems in the urban population of India. National institute of health (NIH) organization observed in a survey that cost of doctor visits, prescription and hospitalization related to gastrointestinal diseases have increased significantly in past one decade.Science daily mentioned in their report that gallbladder stone disease(GSD affects 10 to 25% adults in united states of America. It shows that gallbladder disease affects 10-25% of adults .Although some affected may not have the symptoms. It is the second highest economy consuming gastrointestinal disease annually and results in over population hospitalizations each year.1 The gallbladder is a hollow muscular organ that lies just beneath the right lobe of the http://en.wikipedia org/wiki/Liverliver 2. In adults, the gallbladder measures approximately 8 centimeters (3.1 in) in length and 4 centimeters (1.6 in) in diameter fully distended3.The capacity of gallbladder is 70-100 ml 4. The gallbladder is sac shaped hollow organ, with an opening into the http://en.wikipedia org/wiki/Biliary_treebiliaryhttp://en.wikipedia.org/wiki/Biliary_tree tree through http://en.wikipedia.org/wiki/Cystic_ductcystic duct. Anatomically the gallbladder consists of the neck, body and fundus.The fundus is a rounded end of the organ faces in front of the body at level of the http://en wikipedia.org/wiki/Transpyloric_planetranspylorichttp://en.wikipedia.org/wiki/Transpyloric_plane plane.The body (http://en.wikipedia.org/wiki/Latin languageLatin: corpus) is the middle part of organ, lies in between fundus and neck directly in contact with the inferior surface liver, lying the cystic fossa for gallbladder at the bottom of the liver. The neck is narrowest part of the gland and it is continuous with the http://en wikipedia.org/wiki/Cystic_ductcystic duct. The cystic duct joins with the http://en.wikipedia.org/wiki/Common_hepatic_ductcommon hepatic duct to become the http://en.wikipedia.org/wiki/Common_bile_ductcommon bile duct. At the neck of the gallbladder is a mucosal fold called http://en.wikipedia org/wiki/Hartmann's_pouchHartmann's pouch, where http://en.wikipedia.org/wiki/Gallstonegallstones commonly get trapped. The angle of the gallbladder is positioned between the http://en.wikipedia.org/wiki/Costal_margincostal margin and the lateral margin of the http://en.wikipedia.org/wiki/Rectus abdominis_musclerectus http://en.wikipedia.org/wiki/Rectus_abdominis_muscleabdominishttp://en.wikipedia.org/wiki/Rectus_abdominis_muscle muscle.On the basis of their structure, gallstones can be classified into the following types:1. Cholesterol stones fig (1)http://en.wikipedia.org/wiki CholesterolCholesterol stones vary from light yellow or brown or dark green or chalk white and are oval, usually single, length about 2 to 3 cm, often having a minute, dark, central spot.2. Black Pigment stones fig (2)Pigment stones are small and dark and comprise http://en.wikipedia.org/wiki Bilirubinbilirubin (Insoluble bilirubin pigment polymer) and http://en.wikipedia.org/wiki/Calciumcalcium (calcium phosphate) http://en.wikipedia.org wiki/Salt_%28chemistry%29salts that are found in bile, usually black and multiple.3. Mixed stones (Brown pigment stone) fig (3)In general, major constituent of mixed gallstones is cholesterol it ranges from 20–80% of its total weight (or 30–70% according to the Japanese-
Other common constituents are http://en.wikipedia.org/wiki/Calcium_carbonatecalcium carbonate, http://en.wikipedia.org/wiki/Palmitatepalmitate phosphate, bilirubin and other http://en.wikipedia.org/wiki/Bilin_%28biochemistry%29bile pigments (calcium bilirubinate, calcium palmitate and calcium stearate). Because of their calcium substance, they are often visible by http://en.wikipedia.org/wiki/Radiographyradiographically5. fig fig- (2)
the Greek chol- (bile) + lith- (stone) + iasis- (process) Gallstone disease (GSD) affects about 10 to 20 % of new patients are observed in which 600,000 patients undergo cholecystectomy approximately. Gallstones may be present within the gland for without developing symptoms. Gallstones are formed by cholesterol, bile salts and calcium. Their size ranges from a few millimeters to a few centimeters. Gallstones are formed when bile contains excess amount of cholesterol in it. The consumption of excess cholesterol in diet forms crystals leads to formation of gallstones. People who have genetic background gallstones in their family, people with >25 kg/m2BMI, people with high http://www.netdoctor.co.uk/health_advice/facts/obesity.htmblood cholesterol level, women who are using http://www.netdoctor.co.uk/sex_relationships facts/contraceptivepills.htmcontraceptive pills and people with diseases such as chronic intestinal inflammation (http://www.netdoctor.co.uk/diseases facts/crohnsdisease.htmCrohn's disease and http://www.netdoctor.co.uk/diseases/facts/ulcerativecolitis.htmulcerative colitis) are more prone to develop gall stones. The population based survey from India has shown that prevalence of gall stones was found to be 6.1 %. Incidents among and among females was 9.2%.as per NCRP (2000) .3% male and .9% female are affected by gallstones in India. This calls for active newly arising disease6. Cholesterol gallstone disease, one of the commonest digestive diseases in India, it is induced by disparity in cholesterol metabolism, which involves hepatic biosynthesis, intestinal absorption and biliary output of cholesterol, and its translation in to bile acids Following metabolic syndromes (e.g., obesity,dyslipidemia, type 2 diabetes and hyperinsulinemia) are also well-known risk factors for gallstone formation, suggesting the existence of interplay between common pathophysiological pathways influenced by insulin resistance, genetic and environmental factors. Cholesterol gallstones formation may be enhanced by the abnormal expression of a set of the genes that affect homeostasis of cholesterol metabolism and storage leads to insulin resistance. Additionally, epigenetic mechanisms (mainly DNA methylation, histone acetylation deacetylation, and noncoding microRNAs) may amend gene expression in the absence of an altered DNA sequence, in response to different lithogenic environmental stimuli, such as diet, lifestyle, pollutants, also occurring gestational period before birth.7 Cholesterol gallstone disease is one of the most prevalent and most costly digestive diseases requiring hospital admission, since its prevalence ranges from8-10% in Indian population and united states population .Expenses for treatment of gallstone disease exceeded $4 billion in facility charges in 2004 in the United raised to $6.5 billion when surgical complications occur9.The growth and formation of cholesterol gallstones, which accounts for 75% of

gallstones in westernized countries10 are secondary to abnormal cholesterol homeostasis11. Following metabolic syndromes are the main risk for formation cholesterol gallstone (e.g., obesity, type 2 diabetes, dyslipidemia, and hyperinsulinemia) are also well-known components of the metabolic syndrome supporting the hypothesis that gallstone disease is just another component of the metabolic syndrome. Due to the high prevalence of the metabolic syndromes, it has been advised that the phenotype of cholesterol gallstones may result from the collaboration between insulin resistance, genetic factors, and a number of environmental factors.12 Prevalence- In many standard textbooks of Medicine and Gastroenterology published from the West, India is included along with countries with a low incidence of gallstones. The prevalence of gallstone disease varies in different parts of India. Malhotra as early as in 1966 conducted an epidemiological study in Indian railway employees and showed that North had seven times higher prevalence of gallstones compared to South Indian employees. In northern states including Kashmir where good epidemiological studies have been performed, a very high and increasing prevalence were reported (13,14). Khuroo from Kashmir reported a prevalence of and women 9.6%) the prevalence increasing progressively to reach a peak in the sixth decade (15). The prevalence rate is significantly multiparous women. There was no correlation with diet, obesity or socioeconomic status 16.The identification of large number of cases of cancer of the gallbladder is in agreement with the high prevalence of gallstones observed 17. The pathogenesis of gallbladder cancer albeit only in a number of patients with gallstone disease is not well understood. A high concentration of glucourso deoxycholate and the duration of cholelithiasis are postulated. A recent prospective study has shown however that the development of gallbladder cancer in patients with gallstones followed over decades remains a rare event 18 .Migrants from India to the UK, Australia, fuji, Kuwait and Singapore have a higher incidence of gallbladder cancer as compared to native populations of the areas 19. It is interesting to note the association of gallstones, carrier state and gallbladder carcinoma 20. Sharma and associates from banaras Hindu University studied the association in 390 patients. There about 84 times higher risk of developing gallbladder carcinoma in patients who were bile culture positive for typhoid bacilli 21.The role prophylactic cholecystectomy in patients (with gallstones) from high risk areas for gallbladder cancer is not clear, but should be individualized factors like age, geography, race, size of stone help to identify suitable cases for prophylactic cholecystectomy. Many other studies from Chandigarh and New Delhi confirm the high number of gallstone disease in North India (22).A different picture arises from data available from south India Jayanthi et al (23) reported that mixed and pigment stones were more common than cholesterol stones in Tamilnadu. They found no correlation demographic features or social customs. An interesting observation that needs confirmation is an association with high consumption of tamarind. There is no scientific explanation for this observation. The overall prevalence of gallstones in Tamilnadu appears to be lower than in the North. However cholecystectomy once an extremely uncommon surgery in south India has become very frequent reflecting either a real increase in the prevalence of the disease, better diagnosis because of ease of detecting stones by abdominal ultrasound or the availability of AP cholecystectomy. 5REVIEW OF LITERATURE:Gallstones formation is the most common disorder of the biliary tree and it is unusual for the gall bladder to be diseased in the absence of gallstones.Risk factors-Female gender, consumption of high fatty diet, usage of contraceptive pills, fecundity, and genetic history for gallstone disease are strongly accompanying with the formation of cholesterol gallstones 24. Obesity25, as well type 2 diabetes contributing to syndrome26 such as dyslipidemia (in particular hyperlipoproteinemia27 type IV with hypertriglyceridemia and low HDL cholesterol), hyperinsulinemia insulinresistence28 or overt type 2 diabetes are risk factors for the development of gallstones, itself supposed to be acomplication of the syndrome. Oestrogen treatment enhances the risk, both in women when used for anticonception or hormone-replacement and in men who are under treatment of prostatic cancer with oestrogen harmone therapy29. Among specific dietary factors, short-time high cholesterol as well as high carbohydrate diets were associated with increased risk for gallstones30, and in highly prevalent areas, the intake of legume while unsaturated fats coffee and moderate consumption of alcohol31 seem to reduce the risk. Also physical activity of gallstone disease patient was found to decrease the risk for symptomatic gallstone disease both for men and women32 and independent of weight reduction. On the other hand, rapid weight loss and weight gain33 strongly increase the risk of development of gallstones. Thus, weight reductions process should not exceed 1.5 kg per week34 .Fibrates, for the treatment of dyslipidemia, interfere with cholesterol and bile acid synthesis and increase cholesterol secretion into bile35 However, in contrast to the prototype Clofibrate, during treatment with newer fibrates only a relatively small percentage do actually develop gallstones Pathology-Gallstones are conveniently classified into cholesterol, pigment and mixed stones. Gallstones contain varying quantities of calcium salts including calcium bilirubinate, carbonate, phosphate & palpitate acting as a radio opaque substabce37. 6ROLE OF GENDER AND SEX HORMONEDwivedi et al. (2014) 38 in their study mentioned that women are propably at increased risk because estrogen stimulates the liver to remove from blood and divert it in to the bile An increased risk of gallbladder stone was seen with postmenopausal status because use of hormone replacement therapy (HRT) has more risk for gallstone formation. Narjis hadi et al. (2012) 39 in his study mentioned that incidence of this disease was be higher in females (86%) than in males (14%) the ratio of female to male was 5:1 in his study. Hassan H. Yousif (2011)40 gallstone operated patient. He has observed incidence of gallstone disease in female was more. In his study 132 (88%) patients were patients were males. Hamid .H. Sarhan (2009)41 In their study of Tikrit population he has mentioned that 80% of patient population in were female. Only 90% were males. He also mentioned that female : male ratio was 4% Simona tirziu et al. (2008)42 stone in females is more compared with males. In his study, he has noticed out of 109 patients of gallstone disease 95 were females, were males. Hart AR et al. (2008) 43 The mechanism for the increased risk of gallstone disease in women is probably related as parity, oral contraceptive use and hormone replacement therapy (HRT) all increase the risk of gallstone disease. Ko C.W et al. (2005)44 in their study of gall stone disease in pregnant women. They have concluded that no.of pregnancy play role in the formation of gallstones (lithogenic risk factor). 7Michael W.et al (2001)45 from his study He has observed the incidence of gall stone disease is more in females. He also explain the increased frequency of gallstone in women as an effect of female sex hormones on hepatic function, bile secretion and gallbladder function. According to the Leung J.W (1989)46 study that gall bladder disease like gall stone and other are more common in females at all ages. Although the female: male proportion increase slightly with age. ROLE OF AGE –Dwivedi shipra et al. (2014)47 in her study observed that gallstone disease was postemenopausal women (75%) age between 40-60 years and she also mentioned increased risk of gallbladder stone was seen with because use of hormone replacement therapy (HRT) has more risk for gall stoneIn their study Rajani Sharma (2013) 48observed the higher number gallstone cases were seen in the age between 20-30 and 40-60 years, age around menarche and menopause respectively. ROLE OF LIPID sanikidze et al. (2014)49 in their investigations showed a significant elevation in both high density lipoprotein (HDL) and low density LDL) in the patient blood with gallstone when compared with controls (without gallstone). With this investigations they have concluded the important role of lipid metabolism disorders in the formation of the cholesterol gallstones. 8Dwivedi shipra et al. (2014)50 In her of population she has noticed statistically significant serum high levels of total cholesterol (TC). trigcerides (TG) , low density lipoproteins(LDL

very low density lipoproteins (VLDL).In case group with gallstone disease the then the control group without gallstone disease. He also mentioned that the concentration of high density lipoprotein(HDL) is significantly low in case group when compare with control group.Rajaa et al mentioned that concentration of serum total cholesterol ,LDL,VLDL,TG and fasting serum glucose were significantly higher , while the levels of HDL were significantly lower in the gallstone patients in comparison with healthy control group. He also mentioned there was significant positive correlation (r=0.53,P<0.05)between serum VLDL and TG in patients with cholelithiasis. Cholelithiasis is associated with hyperglyceridemia.According Virupaksha h.s et al. (2011)52 study shown there was a significant increase in total cholesterol LDL cholesterol and an significant decrease in cholesterol in cholelithiasis patients as compared to control subjects. sen wise analysis of the HDL cholesterol has revealed significant in female patients as compared to male patient. Naseem aslam channa et al. (2010) 53study demonstrate that elevated serum total cholesterol, cholesterol, LDL cholesterol, triglycerol, and decreased levels of HDL cholesterol seem to play major contributing role in the pathogenesis gallstones in female up to 45years with more than three children. Zak A et al. (2007) 54indicate that medications used to treat lipidiemia may be value in the prevention and treatment of cholelithiasis. ROLE OF PARITY- Dwivedi Shipra et al. (2014) 55in her study she has with more than 3child birth bave more risk to develop gallstone when compared with women less than 3 child birth in her study she has noticed percentage of women more than 3 children was 60% when compared with women less than 3 child birth was only 40%. 9Kumar concluded the increased number of pregnancies is associated with an increased risk of gallstones believe that the progesterone component rather than the estrogen is responsible for the changes in biliary lipids bile is more saturated during the second and third trimetter of pregnancy.ROLE OF CONTRACEPTIVE USE- According to Dwivedi shipra et al. (2014)57 study women with contraceptive usage has more risk to develop the gall stone when compare with women without contraceptive usage and she also mentioned no. of women with contraceptive usage was 74.3% (52/80) and without contraceptive usage was only 25.7% (18/80) in study group.Chaurasia, et al. (2004)58 observed the use of oral contraceptive also increased risk of gallbladder disease. Oral contraceptive therapy may be associated with an increase in cholesterol saturation that will lead to formation of gallstones.ROLE OF OBESITY (BMI)-Jebb SA, Prentice AM et al. (2001)59 Observed that Increasing obesity is a risk factor for gallstone disease although the mechanism is unknown and data from European studies is minimal. The World Health Organisation defines obesity as “abnormal or excessive fat accumulation that may impair health”.65 Body mass index (BMI; expressed in kg/m2) is often used as a measure of obesity and gives an estimate of relative weight for height. The World Health Organisation categories BMI as follows; BMI 20-25 kg/m2 “normal”, 25-<30 kg/m2 “overweight kg/m2 “obese class I”, 35-<40 kg/m2 “obese class II”, >40 kg/m2 “obese class III” .Gielkens HA, Lam WF et al. (1998)60 Confirmed that Obesity and hyperinsulinaemia also contribute to gallstone formation by causing gallbladder hypomotility which promotes cholesterol crystal aggregation 10Vimal bhandari et al. (2002)61 observed that the mean BMI was statistically higher in case group (27.50.71) than control group (24.310.32kg/m2, and BMI ratio were also associated with occurrence of cholelithiasis. Jacson AS,et al. (2002)62 in his study mentioned that BMI, women have significantly greater amount of total body fat than do men throughout the entire adult life span. Acalovschi M (2001)63 in his study concluded there was a striking monotonic increase in gallstone risk with obesity was observed. The annual gallstone incidence rose parallel with the BMI, up to year in morbidly obese. Further studies confirmed that men's chances of having gallstones are increased by having an abdominal distribution of body fat, leading to the conclusion that obesity represents a risk for gallstones both through total body fat mass and through fat regional distribution Kodama, Kono S et al (1999)64 in their cross- sectional study concluded that BMI was significantly associated with an increased risk of gallstones. WHR also showed a significant positive association. When BMI and WHR were mutually adjusted for, both of the obesity indices tended to be associated positively with prevalent gallstones. These findings indicate that obesity is associated with increased gallstone risk in men.Tandon RK et al. (1996)65 observed that high BMI (>22) correlated with a higher incidence of gall stones. ALCOHOL CONSUMPTION-Saluja AK, Probert CS observed that alcohol may prevent the development of gallstones by several biological mechanisms. Alcohol stimulates cholecystokinin release 116 therefore gut motility117 which prevents biliary stasis and cholesterol crystal aggregation. 11Gaziano JM, Buring JE,et al. (1993)67 mentioned that alcohol intake also increases HDL levels118-120 by reducing cholesteryl ester transfer protein (CETP) activity which prevents the conversion of HDL into LDL.121 HDL is then metabolised to primary bile acids which help to solubilise biliary cholesterol. Alcohol may also increase lecithin cholesterol acyl transferase (LCAT) activity (an enzyme that converts free cholesterol into cholesteryl ester), which further increases HDL.ROLE OF RAPID WEIGHT LOSS- Iglezias Brandao de Oliveira C, Adami Chaim E et al.(2003)68 mentioned that Patients undergoing bariatric surgery are particularly prone to gallstone formation (prevalence pre-operatively of 21.6% vs 1 year postoperative incidence of 52.8%) which is believed to be induced lack of gallbladder stimulation and hence biliary stasis. Decreased calorie intake and rapid bowel transit alter gut hormone secretion, with reduced cholecystokinin release, leading to gallbladder hypomotility.Yang H,Petersen GM et al.(1992) 69observed that weight reduction in obesity is associated with gall stone formation risk. Different studies in the morbidly obese presenting for gastric bypass surgery or after a very low calorie diet showed the occurrence of sludge and gall stone in 10% to 25% of patients in a few weeks after slimming procedure. ROLE OF DIET- Smeets FW et al. (1994)70 observed that calcium intake seems to be inversely associated with gall stone prevalence. 12Orchard TJ,Laakso M,et al mentioned that long chain saturated fatty acids are known to contribute to raised serum triglycerides,and increase insulin secretion and decrease insulin sensitivity which are mechanisms which promote gallstone disease. Both epidemiological and intervention studies of fatty acid classes suggest that saturated fat worsens insulin sensitivity, while monounsaturated and ω-6 polyunsaturated fats improve it.Raised insulin levels cause gallbladder hypomotility and dyslipidaemia leading to increased biliary cholesterol saturation and mucin production, all of which promote stone formation. In studies on hamsters, saturated fatty acids, and in-particular long chain ones, have been shown to increase cholesterol gallstone formation.ROLE OF SMOKING-Sarin SK, Jorgensen T. et al. (1986;1989)72 observed that cigarette smokers have lower level of HDL in comparison to nonsmokers , a factors for gall stone formation in some studies.ROLE OF SOCIOECONOMIC STATUS-Diehl AK, Rosenthal M et al. (1985)73 analysed that those living in affluent neighbourhoods , with high occupational status, high family incomes , and high educational attainment had lower rates of those lower on the social scale.ROLE OF PHYSICAL ACTIVITY- 13Tran ZV, Kirwan JP, Koffler KH et al. (1993)74 observed that there are several pausible biological mechanisms for how physical activity may prevent gallstone formation. Exercise reduces plasma triglycerides and insulin levels, both of which lead to a lower cholesterol saturation of the bile. Triglycerides also increase bile viscosity by stimulating mucin secretion from gallbladder mucosal cells which promotes the aggregation of cholesterol into microcrystals. Regular exercise raises High-Density Lipoprotein–Cholesterol (HDL-C levels which are inversely associated with gallstone prevalence.HDL-C is a precursor of bile acids which reduce its lithogenicity. Exercise, by increasing cholecystokinin levels, has a prokinetic effect on the gut which stimulates gallbladder contractility and prevents bile stasis. To support the experimental data for a protective effect of exercise, large population based epidemiological studies are required showing that those who exercise are at a lower risk of developing gallstones. Both case-control and prospective cohort studies can be used to investigate this potential

association. However, prospective investigations provide more accurate information on pre-symptomatic physical activity, as this is measured before the development of disease and consequently is not subject to the recall biases inherent in case-control studies. The published cohort studies have reported that higher levels of physical activity reduced the risk of symptomatic gallstones by approximately a third. However, a limitation of all these investigations was that the method for measuring physical activity, namely questionnaires had not been validated against detailed physiological measures of physical activity, including energy expenditure and cardiorespiratory fitness. Similar data on exercise is also needed in European populations to clarify the association and define the magnitude of effect. To address these limitations, physical activity questionnaires assessed against energy expenditure (repeated 4- day heart monitoring) and cardiorespiratory fitness (repeated measures of submaximal oxygen uptake) important since they allow the accurate categorisation of physical activity levels. Demonstrating an association of physical activity with gallstones should lead to it being accurately measured and included in aetiological models of gallstone formation. It would also be important as encouraging increased physical activity levels may help to reduce the numbers developing symptomatic gallstones.ROLE OF GENE - Hepatic cholesterol may be converted to bile acids or secreted directly into the bile via reverse cholesterol transport. In humans, the pool consists of “primary” cholic acid CA) and chenodeoxycholic acid (CDCA) and“secondary” deoxycholic acid and lithocholic acid bile acids. Primary bile acids are synthesized from cholesterol via the classic and alternative hepatic pathways. In contrast to CA, CDCA may reduce the degree of cholesterol bile saturation thus decreasing gallstone formation. In the classic pathway, cholesterol 7 a –hydroxylase (CYP7A1) and microsomal sterol 12 a -hydroxylase (CYP8B1) are rate-limiting for CDCA and CA synthesis, respectively. In addition, mitochondrial sterol 27-hydroxylase (CYP27A1) initiates the alternative pathway in both CA and CDCA synthesis Secondary bile acids are derived from primary bile acids by intestinal bacterial enzymes (LXR) up regulation of CYP7A may promote reverse cholesterol transport from the peripheral tissues to the liver for increased bile acid synthesis .The nuclear hormone farnesoid X receptor (FXR), encoded by NR1H4, induces ABCB11 expression and downregulates CYP7A1. 14The CYP7A1 gene cytochrome P450, family7,subfamilyA polypeptide 1. Involved in lipid metabolism, and bile acid biosynthesis pathway and SNP is -204A>C AND results is percentages of a allele in patients controls were 62.86% and 54.38% (P<0.005) function is rate limiting enzyme of bile salt synthesis.Pullinger et al.75 found a link between a gene defect of cholesterol 7 alpha-hydroxylase (CYP7A1) and cholesterol gallstones associated with hypercholesterolemia resistant to 3-hydroxy methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors in two homozygote males. The homo-zygous deletion mutation in CYP7A1 gene could result in loss of enzyme function. CYP7A1 is a rate-limiting enzyme in the classical pathway for liver bile salt synthesis. As such, decreased bile salts in bile may lead to increased gallstone formation SUMMARY OF REVIEW OF LITERATUREGallstones are a common clinical problem and their formation is complex involving many patho-physiological and biochemical mechanisms. These include cholesterol saturation , aggregation of crystals and stasis of the gallbladder. Many factors can affect these processes including the well established risk factors of: GenderObesity Physical Activity BMIRapid weight loss DietMany nutrients including food groups vitamins and minerals may impact upon stone formation, although the current epidemiological is either limited prospective cohort work or uses less accurate measures of dietary intake.The aim of this study is to assess the effects of life style factors including obesity,physical activity ,diet and alcohol on the risk of developing symptomatic gallstone disease in both genders in a large Indian prospective study. Data from serum lipids will be also analysed to clarify the potential biological mechanism for how lifestyle may affect gallstone formation. 15The CYP7A1 is a rate- limiting enzyme in the classical pathway for liver bile salt synthesis and if it is decreased, bile salts in bile may lead to increased gallstone formation. Confirmation and quantification of potential risk factors associated with symptomatic, gallstones will further enhanced our understanding of gallstone aetiology and it could influence public health policy to prevent biliary stone disease. HYPOTHESIS: Female, fatty, fertile, fair, forty (5f) has association in the formation of gallstone formation. Abnormal lipid profile is associated in the formation of cholesterol gallstone formation.Gallstone formation has association with CYP7A1 gene polymorphism. Low physical activity , rapid weight loss, iron deficiency , high mineral content in the Drinking water play a role in the existence of gallstone serum bilirubin concentration is associated with formation of pigment stones. Normal or high heme iron concentration is reduces the risk of gallstone formation .Anthropometric measurements like waist circumference, hip circumference,waist hip ratio and mid arm circumference shows correlation in the formation of gallstone. 17AIMS AND OBJECTIVES: AIMS:We will estabilish the correlation of socio- demographic findings with existence disease. The correlation of CYP7A1 gene polymorphism with type of gallstone. Correlation of serum lipid profile and heme iron concentration with type of gallstone formation.Association of anthropometric variable like waist, hip circumference and waist hip ratio in gallstone formation. OBJECTIVES
To find out the influence of lipid profile on gallstone formation. To observe the protective role of heme iron in the formation of gallstone. To examine the role of socio-demographic status in gallstone formation. To find out the association of CYP7A1 gene polymorphism in gallstone disease. To examine the role of rapid weight loss, physical activity in the existence of gall stone disease. 18MATERIALS AND METHODS:
study will be conducted at the department of Anatomy and surgery Rama medical college Hospital and Research Centre, Rama university Kanpur.
the study approval of project will be taken from the institution ethical committee. Gallstones will be collected from department of surgery and appropriate examination will be done to asses the type. To define waist hip circumference, waist hip ratio and mid arm circumference will be observed in all patient.Detail history of patient regarding the socio- demographic profile and present and past medical history will be recorded on designed pre tested proforma. AgeGenderWeightHeightArea of subject: Rural/urbanEconomy: below poverty line /above poverty line Literacy: illiterate primary/ secondary/graduate.Parity of women: prime/multi parity.Working women/housewife Alcohol: Yes/NoAddiction : Tobacco chewing : Yes/NoPhysical activity : mild/moderateRapid weight loss in past 6 monthsCoffee or tea drinking habbit: once in a while/regularly.Food style: vegetarian /non vegetarianSmoking yes/no 19Following hematological and biochemical investigation done on subject edta blood.Complete blood picture (CBP).Heme iron concentration.Serum bilirubbin.Lipid profile - serum cholesterol - serum triglycerides. -VLDL -LDL -HDLSerum calcium.Following genetic investigation will be done with subject’s venous blood.MOLECULAR ANALYSISSampling: 5ml of venous blood was drawn from peripheral vein using Di sodium EDTA vaccutainers. All the samples were aliquoted and stored at -700c until tested.The laboratory work was carried out in the central research laboratory Rama medical college, hospital &research centre. All the groups will be analyzed for CYP7A1 (-204), gene SNP –rs3808607 (Homo Sapiens) which is located on chromosome 8. Methodology: 1) Isolation of DNA: DNA will be extracted from whole blood containing EDTA by a standard salting
The reagents required for DNA isolation were; Chemicals used for DNA isolation1 M tris buffer(ph-7.5) 1 M Mgcl2 1 MM Kcl 200.5 M EDTA10% SDS5 Triton X 100EthanolReagentsRBC LYSIS BIFFER:10 mM of tris HCl10 mM of KCL10 mM of Mgcl22 mM of EDTANUCLEUS LYSIS BUFFER:10 mM of tris HCL10mM of KCL
mM of Mgcl22mM of EDTA400 mM of Nacl21All the components will be mixed with sterile double distilled water and the prepared reagents were stored at cProtocol for DNA isolation (salting out Method)Thawed blood samples (5ml) from EDTA vaccutainers were transferred into clean and sterile centrifuge tubes.10ml of RBC lysis buffer was added and the volume made upto 15ml.0.1% of triton X was addedThe centrifuge tubes were incubated at 370c for

minsThe samples were spun at 2000rpm for 15 mins in a cold centrifuge at 40c 21After centrifugation the supernatant was discarded off taking care the pellet.10ml of RBC lysis buffer was again added and vortex mixedThe samples were spun at 2000 rpm for 15minsThe supernatant was discarded off and a white pellet was obtained 1 ml of nucleus lysis buffer was added then vortex mixedAbout 20µl of 10%SDS was added and mixed properlyThe mixture then incubated in a wate bath at 550c for 1 hourAfter incubation,the contents were transferred into 2ml eppendorf tubes.400 µl of 5M Nacl was addedThe contents were spun at 10,000 rpm for 15minsAfter spinning the supernatant was transferred into a new centrifuge tube leaving behind pellet in the eppendorf tubesAbout double the volume if cold absolute ethanol was addedLarge clumps of DNA were obtained by gentle swirling of the tubesThe DNA were scooped with the help of a sterile spatula or a sterile loop,from the centrifuge tubes and transferred into eppendorf tubes200µl of ethanol was addedThe contents were centrifuged and the eppendorf tubes with the DNA were kept for air dryingAfter drying, about150µl of TE buffer was added and the DNA stored at -200c for later use. Concentration and quality assurance of DNA: 22The quality of the DNA was analyzed by agarose gel electrophoresis and the quality assessed by standard spectrophotometer at 260 nm and 280 nm.2) Molecular characterization of ACE gene by Polymerase chain reaction: To determine the CYP7A1 genotype of cases and the control groups, the genomic DNA fragments on the will be amplified by PCR. The condition required for amplification mentioned below:PCR CONDITIONS:Initial denaturation:940c-5minsDenaturation: 940c secsAnnealing:58oc-45secsExtension :720c-45secsCycling condition :30 cyclesFinal extension:720c-7minsHold at 40cPCR reaction mix (working concentration) The working conditions for PCR reaction mixture were;10 x PCR Buffer:1XdNTPs:200µMForward primerr:30pMReverse primer:30pMTaq polymerase:1.5UTemplate DNA:50µgDistilled water :to make up the volume 23 Primers for CYP7A1 polymirphism used in this study: The flanking primer sequences as reported by rigat et al,1992 were;Forward primer: 5'CAGAGCATGGACAGGGAGCAG 3'Rewarse Primer: 5'GCAACTCCTCATGGCTGAGGTT 3'Primers will be
obtained from Banglore Genei and will be reconstituted with sterile double distilled water based on the manufacturer's instruction. Agarose Gel ElectrophoresisOnce the amplification will be obtained, they were subjected to 2% agarose gel electrophoresis with Ethidium bromide and the were visualized under UV light. the requirements for electrophoresis were;Requirements for electrophoresisPurified Agarose 50x TAE bufferEthidium bromideBromophenol Blue Composition of 50 X TAE Buffer (1000Ml)Tris Base :242 gAcetic acid :57.5mlEDTA:18.6gpH:7.2 24 Procedure prepared ,by mixing 2 g of agarose in 100ml of bufferThe contents were boiled completely till a clear solution was obtainedAbout 100 bromide was added and mixed properlyAgarose was casted into specific rays and allowed to solidifySpecific combs were used to make sample wells on the gelThe PCR products were mixed with bromophenol blue12µl of DNA ladder was loaded into the first wellThe samples were then loaded into the gels and run at 100vAfter electrophoresis, The PCR products were visualized under UV light. In the case of Deletion (D allele) and Insertion (I allele), a bp and a 490 bp fragment were obtained respectively and comparison was done with a DNA ladder on the first well Following anthropometric done on subjects:Waist circumference Hip circumferenceWaist/ hip ratioMid arm circumference Standard investigation done on gallstone to find out the TypeSizeNumber of gallstone. 25 STATISTICAL ANALYSIS:Sample size has been calculated in order to control type I & type II error. Assuming a power 80% and 95% significance level the sample size has been calculated using this formula: 2 p- Incidence of p) (P1-p2)2 or d2 – Is the difference which we want to detect at a specified power & level of confidence. Zβ - power of statistical test we be minimum 80% for which is Zβ is 0.84.Zα/2 –is the level of confidence we have choosen 95% confidence in this Zα/2=1.96.When P indicates incidence of the clinical condition e.g.: gallstone disease.Following the literature the incidence of gallstone disease has been assumed between The calculated minimum sample size for our study is 88.The calculated minimum sample size for control group is 88.In order to control loss of follow up and manual errors, we finalised the sample size 150 for each group. Data will be collected and entered in MS excel worksheets and results will analysed with appropriate statistical tools like, tests of significance, logistic regression analysis etc using SPSS version software. 26METHOD OF SAMPLING:Gallstones will be collected immediately after surgery. These are identified by I.P number of patient and stored for investigation.2 groups are madeGroup-I: Control group (Healthy person without gallstone disease screaned by USG scaning)Group-II: gallstone disease patient INCLUSION CRITERIA:Patients who had diagnosed as gall stone diseased patients by ultra sound scaning (USG).Patients who got operated for removal of gallstones who are willing to participate in the study. EXCLUSION CRITERIA: 1. Patients who are not willing

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